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HUMAN PLEURAL FLUID IS A POTENT GROWTH MEDIUM FOR BACTERIA ESPECIALLY STREPTOCOCCUS PNEUMONIAE

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Aim: Empyema can complicate pneumonia, and is defined by the presence of bacteria and/or pus in pleural effusions. *Streptococcus pneumoniae* is one of the commonest bacteria isolated from empyema fluid. It is believed that bacteria are shed from infected lung/pleural tissue into the fluid. However, the subsequent biological interactions between bacteria and pleural fluid (and its cellular contents) have not been studied.

We hypothesize that pleural fluid presents a rich culture medium that facilitates bacterial growth. This study aims to determine whether pleural fluid, with/without its cellular content, enhances proliferation of *S. pneumoniae* and other common pneumonia/empyema pathogens.

Methods: Invasive *S. pneumonia* strains (n = 21) isolated from empyema or blood and laboratory reference strains (n = 5) were tested, alongside 14 strains of randomly-selected pathogens (*Staphylococcus aureus, Pseudomonas aeruginosa, S. anginosis, Escherichia coli* and *Klebsiella* species).

Pleural fluids (all culture negative) collected aseptically from patients with pleural malignancies were used as media for bacteria culture. Matched pairs of pleural fluid or its supernatant (without cellular material) were inoculated with 1×10^6CFU/mL of bacteria and incubated at 37°C. Manual counts of serial dilutions were performed to quantify bacterial concentrations.

Results: All 26 strains of *S. pneumoniae* proliferated rapidly, by an average of >4500 fold over baseline, in all (n = 11) effusion samples tested (range of fold increase from 563 to 12351) after 24 hours. The bacteria did not grow in saline controls. The supernatant supports the same potent proliferative effect as un-centrifuged pleural fluid, $p = 0.984$. The growth of other respiratory pathogens in pleural fluid was variable. Half of 14 other bacteria tested did not proliferate in the pleural fluids (n = 7) samples.

Conclusion: Pleural fluid can provide a rich medium for proliferation of some bacteria especially *S. pneumoniae*. Proliferation is independent of the presence of cellular content of the fluid.

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EARLY ONSET SEVERE DISEASE AND PREMATURE MORTALITY IN INDIGENOUS AUSTRALIANS WITH NON-CYSTIC FIBROSIS BRONCHIECTASIS

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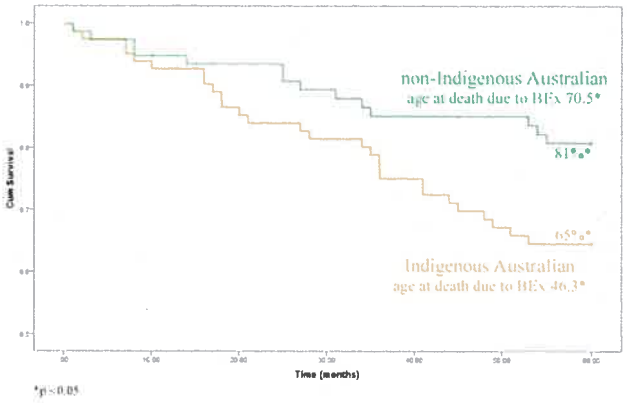
Aim: To describe the natural history of adult non-cystic fibrosis bronchiectasis (non-CF BEx) and identify risk factors associated with premature mortality within a cohort of Indigenous and non-Indigenous Australians.

Methods: A retrospective cohort study of Indigenous and non-Indigenous Australians with non-CF bronchiectasis from January 1 2009 until January 15 2014. Five-year survival was determined and multivariate Cox proportional hazard modelling used to identify independent predictors of mortality.

Results: 85 Indigenous (all Aboriginal Australian) and 79 non-Indigenous Australian adults were included. Aboriginal Australian non-CF BEx patients were younger (mean age 43.7 ± 12.3, SD versus mean age 59.4 ± 17.5 in non-Indigenous Australians, $p < 0.001$) and had more severely impaired lung function with lower FEV1% (median 30.0 (IQR 20.0–37.0) compared with median 48.0 (39.0–81.0, $p < 0.001$) and FVC % predicted (median 41.0 (31.5–48.0) compared with 65.0 (48.0–93.0, $p < 0.001$). Five-year respiratory-related survival in Aboriginal Australians was poorer (log rank, $p = 0.029$) (see Figure).



Factors independently associated with poorer respiratory-related survival were; being Aboriginal Australian (HR 4.12, 95% CI 1.71–9.94, $p = 0.002$), increasing age (HR 1.04/year, 95% CI 1.01–1.07, $p = 0.012$), male gender (HR 2.75, 95% CI 1.32–5.76, $p = 0.007$) and more lobes affected (HR 1.31/additional lobe, 95% CI 1.05–1.64, $p = 0.018$).

Conclusion: Indigenous Australians with non-CF bronchiectasis experience severe early onset non-CF bronchiectasis that ultimately leads to premature respiratory-related mortality compared to non-Indigenous Australians. This has implications for both prevention and management.

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